Table Nerve conduction studies

Episode	Time from onset (days)	Motor conduction: Median Latency (ms)	Median Amplitude (mV)	Median Velocity (m/s)	Ulnar Latency (ms)	Ulnar Amplitude (mV)	Ulnar Velocity (m/s)	Common peroneal Latency (ms)	Common peroneal Amplitude (mV)	Common peroneal Velocity (m/s)	Sensory conduction: Median Amplitude (μV)	Median Velocity (m/s)	Ulnar Amplitude (μV)	Ulnar Velocity (m/s)	Sural Amplitude (μV)	Sural Velocity (m/s)
1	10	12·8	8·0	29·4	7·5	10·0	31·6	NR	NR	NR	NR	NR	NR	NR	NR	NR
	120	4·4	12·0	52·2	3·4	14·0	50·2	4·6	11·1	48·4	10·0	51·4	9·2	55·4	14·2	50·5
2	9	17·5	5·5	26·6	6·5	7·0	33·7	NR	NR	NR	NR	NR	NR	NR	NR	NR
	60	6·0	10·0	47·0	3·6	11·0	53·6	6·5	7·0	33·8	NR	NR	NR	NR	NR	NR
3	14	10·4	7·6	21·8	6·8	2·3	27·2	7·2	4·1	31·8	4·0	40·6	6·0	31·2	NR	NR
	45	5·4	13·0	40·0	4·0	10·0	50·8	25·5	9·0	41·5	7·0	51·7	10·0	56·6	NR	NR
Controls (n = 28)																
Range		2·8–4·2	6·5–22·0	44·6–61·7	2·0–3·5	10·6–20·0	48·9–66·7	3·4–6·2	4·0–14·4	36·8–52·1	7·0–36	45·0–70·7	5·0–30·0	45·1–71·4	13–60	37·3–57·4

NR = no response.

hospital. On follow up after four months, he showed complete recovery both clinically and electrophysiologically (table).

In September 1991, he had a severe attack of acute filariasis. Three weeks later he developed a flaccid, areflexic tetraparesis with distal sensory loss. After three days he developed tetraplegia (MRC grade 0) with bilateral lower motor neuron facial weakness, dysphagia, and nasal regurgitation. Gag reflex was absent. At the same time he had respiratory difficulty and required assisted ventilation for five days. The CSF on the ninth day was acellular with an elevated protein content (380 mg/dl) and did not show any microfilariae. Electrophysiological studies were suggestive of primary demyelinating radiculoneuropathy (table). He was treated with intravenous dexamethasone 8 mg eight hourly, and with supportive treatment. When he was stable, he was put on oral prednisolone 40 mg/day in divided doses. At the time of discharge from the hospital after two months, he had largely regained limb function but was left with residual, bilateral lower motor neuron facial weakness and minimal distal weakness in the limbs (MRC grade 4^+).

The third episode followed a severe attack of acute filariasis in September 1992. Three weeks later he again presented with flaccid, areflexic tetraparesis (MRC grade 4/5 proximally and 3/5 distally) with bilateral lower motor neuron facial weakness, dysphagia, and nasal regurgitation. Gag reflex was diminished. There was no evidence of respiratory involvement. The CSF on the 12th day again showed acellular response with elevated protein content (640 mg/dl) without any evidence of microfilariae. Nerve conduction studies were suggestive of demyelinating radiculoneuropathy (table 1). He showed remarkable recovery after physiotherapy for six weeks, and recovered completely within three months.

The findings of leucocytosis, eosinophilia, the presence of numerous microfilariae in peripheral blood smears, raised ESR, and serum IgE levels were also confirmed during the second and third attacks of acute filariasis.

During the three admissions the following investigations showed no abnormalities in the blood: red cell count, haematocrit, urea, creatinine, electrolytes, glucose, liver and kidney function tests, Venereal Disease Research Laboratory test, rheumatoid factor, antinuclear antibodies and LE cell tests, serum complements and immune complex levels, serological tests for hepatitis B surface antigen and antibody, and for human immunodeficiency viral antibodies.

In the CSF, microscopic examination of stained specimen (Gram) and cultures for acid-fast bacilli, bacteria, and fungi were normal.

The table shows the results of nerve conduction studies during the acute attack and following recovery. During the attack, no F wave could be elicited although normal or near normal F wave latencies were obtained after recovery. EMG showed no evidence of denervation at any stage, in all three episodes.

Meningitis, encephalitis, encephalomyelitis, cerebellar ataxia, seizures, intracranial hypertension, behaviour disturbances, movement disorders, or spinal cord compression are the known neurological manifestations of filariasis.[1-5] Extravascular cerebral and meningeal spread, immune allergic reactions, and direct compression of the spinal cord are the mechanisms of nervous insult.[3]

Our patient had recurrent Guilain-Barré syndrome, developing severe neurological deficit requiring, on one occasion, mechanical ventilation. All episodes were preceded by a severe attack of acute filariasis. Despite the severity of motor and sensory dysfunction with supportive treatment there was always almost complete recovery. The diagnosis of filariasis was strongly supported by the history of typical inflammatory episodes, the presence of tender inguinal lymphadenopathy and lymphoedema of the right leg, microfilariae and eosinophilia in the blood smear, and a raised serum IgE level.

It is possible that recurrent Guillain-Barré syndrome was a delayed manifestation of acute filariasis, although it is difficult to establish a causal relationship between the two conditions.

B BHATIA
S MISRA
Department of Neurology, Laboratory of Clinical Neurophysiology. Institute of Medical Sciences, Banaras Hindu University, Varanasi - 221 005, India

1 Dumas M, Girard PL. Filariasis of the nervous system. In: Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology, part III. Infections of the nervous system.* Amsterdam: North-Holland 1978;35:161–73.
2 Bada JL, Fernandez-Nogues F, Cerde E, Rufi G. Neurological manifestations in a patient with filariasis. *Br Med J* 1976;2:978–79.
3 Bharucha NE, Bhabha SK, Bharucha EP. Fungal and parasitic infections. In: Bradley WG, *et al*, eds. *Neurology in clinical practice.* Butterworth-Heinemann 1991;II:1120–31.
4 Orihel TC. Cerebral filariasis in Rhodesia. A zoonotic infection. *Am J Trop Med Hyg* 1973;22:596–99.
5 Van Bogaert L, Dubois A, Janssens PG, Rader-Mecker J, Tverdy G, Wanson M. Encephalitis in Loa loa filariasis. *J Neurol Neurosurg Psychiatry* 1955;18:103–19.
6 Padmini R, Maheshwari MC. Vivax malaria complicated by peripheral neuropathy with electrophysiological studies. *J Assoc Physicians India* 1980;28:152–6.

Hypokalaemia mimicking Guillain-Barré syndrome

The rapid onset of areflexic weakness is usually due to Guillain-Barré syndrome, but other important causes include metabolic derangements, the periodic paralyses, botulism and poliomyelitis. We report a case mimicking Guillain-Barré syndrome clinically, and initially electrophysiologically, due to the hypokalaemia of renal tubular acidosis, and we document serial nerve conduction studies during recovery.

A 48 year old woman was fit until 1983 when she embarked upon a severe diet involving laxative abuse, losing 50% of her body mass in three months. Subsequently she developed mild generalised weakness and was found to be hypokalaemic. The weakness improved with potassium replacement, but she was noted to have renal impairment with plasma creatinine of 250 μmol/l. She then remained well for eight years until January 1991 when she presented with a five day history of progressive limb weakness and respiratory difficulty.

On admission there was global reduction in peripheral tone and muscle strength (distal 3/5, proximal 2/5 MRC scale) with absent leg reflexes and flexor plantar responses. Sensation was normal. There was bilateral facial weakness and examination of the eye movements showed hypometric saccades in the horizontal and vertical planes. Systemic examination was unremarkable apart from a low vital capacity of 1·7 litres, supine. On admission her weakness worsened and vital capacity declined to 1 litre; artificial ventilation was therefore started.

Initial investigations revealed raised plasma levels of sodium (153 mmol/l) and decreased levels of potassium (1·9 mmol/l), with abnormal renal function (urea 22 mmol/l, creatinine 333 μmol/l). Glucose, calcium, magnesium, and creatine phosphokinase were normal. A full blood count showed a raised white count of 22 000/ml (90% neutrophils). Plasma osmolality was 300 mosm/kg and urine 289 mosm/kg consistent with an element of nephrogenic diabetes insipidus. Arterial blood gases revealed a metabolic acidosis (pH 7·1, bicarbonate 6 mmol/l, base excess −24 mmol/l, pO_2 24 and pCO_2 3·1 kPa on inspired 30% oxygen). Urine analysis showed a pH persistently below 6·5 with a sodium of 105 mmol/l and potassium of 24 mmol/l. CSF analysis was normal.

The table shows the results of nerve

Table Nerve Conduction Studies

	Day 2	Day 3	Day 7	Normal[8]
Mean serum K (mmol/l)	1·9	3·2	4·0	3·5–5·0
Sensory action potential amplitude (μ V)/peak latency (ms)				
Right median F2-wrist	20/4·0	20/3·4	20/2·8	> 8
Right ulnar F5-wrist	12/3·8	9/2·9	12/1·9	> 7
Right sural	8/5·6	18/3·6	13/3·7	> 5
Compound muscle action potential distal/proximal stimulation (mV)				
Right median (SE APB)	1·5/0·6	4·0/3·8	NT	> 3·5
Right ulnar (SE ADM)	0·7/0·6	5·0/4·6	4·5/3·8	> 5
Right lateral popliteal (SE EDB)	1·2/1·2	0·8/0·8	1·0/0·9	> 2
F wave latency (ms)				
Right median (wrist)	35	32	NT	< 31
Right ulnar (wrist)	44	30	29	< 32
Motor conduction velocity (m/s)				
Right median (forearm)	46	49	NT	> 48
Right ulnar (forearm)	42	58	55	> 50
Right lat. popliteal	38	43	47	> 39
Distal motor latency (ms)				
Median	5·8	3·9	NT	< 4·8
Ulnar	3·5	3·2	2·4	< 3·5
Lateral popliteal	5·0	6·0	4·1	< 6·5

SAP = sensory action potential; CMAP = compound muscle action potential; APB = abductor pollicis brevis; ADM = abductor digiti minimi; EDB = extensor digitorum brevis, MCV = motor conduction velocity; DML = distal motor latency; NT = not tested.

conduction studies performed in the intensive care unit on days 2, 3, and 7. The first study showed a marked reduction in the amplitude of surface recorded compound muscle action potentials (CAMPs) to distal stimulation of the median, ulnar, and lateral popliteal nerves, with slight prolongation of distal motor latencies and delay and paucity of F waves. Motor conduction velocities were slightly reduced but sensory action potentials were of normal amplitude. Electromyography with a concentric needle electrode in the tibialis anterior revealed no spontaneous activity, but no motor units were seen under voluntary control and there was only a small visible muscle twitch to nerve stimulation (the patient was mildly sedated for ventilation, but neuromuscular blocking drugs were not administered). An electrocardiogram on admission was normal.

Initial treatment consisted of intravenous potassium and fluids with parenteral antibiotics. As serum potassium rose, her strength improved and leg reflexes returned. Between days 2 and 3, proximal muscle power increased from grade 2/5 to 4/5, and distal power increased from grade 3/5 to 4+/5 on the MRC scale. This was associated with a commensurate improvement in the size of CMAPs, and a decrease in distal and F wave latencies to normal as serum potassium levels rose from 1·9 to 3·2 mmol/l (table 1). A repeat EMG of upper and lower limb muscles on day 3 showed a severely reduced interference pattern with motor units firing irregularly at 10–20 Hz at 1–2 mV. Occasional units were of spiky configuration but most were of normal form and amplitude.

The metabolic acidosis and polyuria persisted and potassium remained at 3·3 mmol/l, so parenteral bicarbonate was given, with 5% dextrose and spironolactone for secondary hyperaldosteronism. After three days she was given oral bicarbonate (3·6 g $NaHCO_3$ and 17 g $KHCO_3$). This therapy was associated with a progressive improvement in her condition and seven days after admission her muscle power and respiratory function had fully recovered. Nerve conduction studies at this stage had also returned to normal. Her electrolytes and polyuria gradually improved and by discharge her potassium was 4·2 mmol/l, creatinine 176 μmol/l, and she was passing three litres of urine a day.

The most striking initial electrophysiological abnormality was a severe and generalised reduction in CMAP amplitudes; this, in association with modest and mainly distal slowing of motor and sensory nerve conduction and some F wave latency prolongation, was compatible with, although not specific for, the acute phase of predominantly distal Guillain–Barré syndrome. The rapid recovery of CMAP amplitudes in association with the resolution of hypokalaemia and improved muscle power, however, suggests that the low potassium was responsible for the clinical and electrophysiological abnormalities. The recognised electrophysiological features of hypokalaemia, as seen in periodic paralysis, are related to muscle membrane inexcitability[1 2] rather than nerve involvement.

Nevertheless, the exact pathophysiological mechanism of muscle weakness in these conditions is not well established; serum potassium concentration is not consistently related to the occurrence or degree of weakness, and electromyography late in the course of hypokalaemic periodic paralysis may show both neurogenic and myopathic features.[3] In the present case, the abnormalities of motor conduction and electromyography could be explained by inexcitability of muscle fibres, especially those supplied by large, fast conducting myelinated nerve fibres. Although temperature effects may have contributed to the prolonged distal latencies in the initial intensive care unit study, cooling causes an increase in CMAP amplitude and therefore could not be responsible for the most prominent neurophysiological abnormality.[4] Furthermore, conduction block in distal motor nerve fibres as part of Guillain–Barré syndrome would tend to decrease with cooling, rather than the reverse.

A further interesting feature of this case is the apparent differential response of skeletal and cardiac muscle to hypokalaemia, as the ECG on admission was normal. Hypometric saccades in the horizontal and vertical planes were recognised in our patient as a surprising finding for the syndrome, where ocular involvement, when present, is usually manifest as external ophthalmoplegia with slowed saccades.[5]

There have been a number of published reports of hypokalaemic weakness resembling Guillain–Barré syndrome. Causes have included the periodic paralyses, barium toxicity, renal tubular acidosis, and even clay ingestion,[2 6 7] but none have documented serial electrophysiological studies. This case reinforces the need for awareness of the effects of electrolytes, in particular potassium, calcium and magnesium, in both the clinical and electrophysiological assessment of weak patients.

We are grateful to Dr J A Morgan-Hughes for permission to report this patient.

T T WARNER
S MOSSMAN
N M F MURRAY
The National Hospital for Neurology and Neurosurgery, Queen Square, London, United Kingdom

1 Shy MG, Wanko T, Rowley PT, Engel AE. Studies in familial periodic paralysis. *Exp Neurol* 1961;3:53–121.
2 Layzer RB. Periodic paralysis and the sodium-potassium pump. *Ann Neurol* 1982;11: 547–52.
3 Links TP, Zwarts MJ, Wilmink JT, Molenaar WM, Oosterhuis JGH. Permanent muscle weakness in familial hypokalaemic periodic paralysis. *Brain* 1990;113:1873–89.
4 Denys EH. AAEM Minimonograph #14: The influence of temperature in clinical neurophysiology. *Muscle Nerve* 1991;9:795–811.
5 Hughes RAC. *Guillain–Barré Syndrome:* London: Springer-Verlag, 1990:121–140.
6 Shankle R, Keane JR. Acute paralysis from inhaled barium carbonate. *Arch Neurol* 1988;45:579–80.
7 Latiere M, Dumont JC, Olmer M, Francois G. Hypokalaemic quadriplegia and coma in renal tubular acidosis. *Ann Fr Anesth Reanim* 1989;8:133–6.
8 Le Quesne PL. Nerve conduction in clinical practice. In: Licht S, ed. *Electrodiagnosis and electromyography* 3rd ed. III. Licht S. 1971: 419–51.

Polyneuropathy following parathion poisoning

Delayed neurotoxicity is a sequela of poisoning with certain organophosphorus compounds, and correlates with the irreversible inhibition of neuropathy target esterase (previously known as neurotoxic esterase).[1] This enzyme is widely distributed in the nervous system, but its physiological role is not yet known. In humans, the delayed axonal polyneuropathy occurs one to three weeks after intoxication and pyramidal tract signs are often present. The likelihood of organophosphorus compounds producing a delayed polyneuropathy is predicted by their ability to induce a similar syndrome in adult hens and these compounds are called 'neurotoxic'.[2] Recent clinical reports, however, have shown that compounds not usually effective in producing delayed neurotoxicity in adult hens may do so in humans following massive exposure.[3–5] Because the pathophysiology in these intoxications is not yet understood, we present the clinical features of an additional case of ethyl-parathion induced delayed polyneuropathy.

A 23 year old man suffered from depression since the age of 19. He had attempted suicide on three occasions. His admission followed the ingestion of 15g of ethyl-parathion (E605 forte) and the infliction of a gunshot injury to his neck, penetrating the esophagus. Surgical treatment of this injury prevented gastric lavage. Ethyl-parathion serum levels were initially 400 ng/ml, increased to 550 ng/ml at day five and did not begin to decrease until day eight despite 10 haemoperfusions during this time.